Hypertrophic Pyloric Stenosis

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• **Hypertrophic pyloric stenosis, a relatively common condition, is caused by hyperplasia of the musculature of the pylorus. The diagnosis is made by a history of projectile vomiting and failure to gain weight, the observation of gastric peristaltic waves, and the palpation of a pyloric "tumor." A method of palpating this tumor is described in detail. Roentgenological studies are rarely indicated.**

Pylorotomy for treatment of hypertrophic pyloric stenosis was not successful until the development of necessary supporting measures.

Preparation for operation consists of intravenous administration of fluids and electrolytes and sometimes serum or whole blood. The position of the tumor governs the choice between two different incisions. The operative procedure herein described is essentially that devised by Ramstedt many years ago, with modifications to facilitate the procedure.

ACCURATE DATA on the incidence of congenital hypertrophic pyloric stenosis are not available, but it is probably somewhere between 1 in 200 and 1 in 500 births. It occurs about five times as often in males as in females. More than 50 per cent of patients are first-born children, although only 40 per cent of all children are first-born.[5]

PATHOLOGIC AND ETIOLOGIC FEATURES

The so-called "pyloric tumor" is a firm pale swelling of the pyloric end of the stomach, made up of hypertrophy and perhaps also hyperplasia of the musculature, primarily the circular layer. The blunt distal end projects into the duodenum in the manner of the cervix into the vagina. A longitudinal cut section of the mass has a gray-white, quite avascular appearance and a gristly texture. The mucosa is sometimes quite normal, but frequently edema, congestion or even superficial ulceration is present.

The cause of this relatively common condition remains obscure. It would seem that prolonged pylorospasm with work hypertrophy is the most logical explanation. The spasm could be initiated by the central nervous system, by local abnormal innervation,[8] by local erosions or irritation. Supporting this theory is the fact that the "tumor" disappears after pyloromyotomy but remains after gastroenterostomy. Another phenomenon in accord with the theory is the apparent cure in some cases by nonsurgical methods involving the use of antispasmodics. The theory is weakened, however, by the apparent lack of proof as to what brings about the prolonged pylorospasm. Other factors that cannot be readily fitted to such a postulation are the frequency of familial occurrence[1] and the predominance of the disease in males and in first-born.

DIAGNOSIS

Vomiting is the cardinal symptom. It is caused by obstruction at the plyoric canal, which hypertrophy of the musculature has narrowed. In addition, edema of the mucosa, brought about by prolonged forceful propulsion of gastric contents through the narrowed canal, further reduces the lumen. The time needed for development of these changes is probably why vomiting commonly does not start until the third to the sixth week of life, rarely at birth. At onset, vomiting may simply be mild regurgitation; but it gradually becomes more forceful and then almost always projectile in character. The baby may not vomit every feeding but may retain two or three feedings, then vomit the entire residual. An important point is the eagerness with which the infant will immediately take another feeding soon after vomiting, indicating absence of the nausea that is associated with other causes of infantile vomiting. Because the obstruction is in the pylorus, the vomitus is not bile-stained. The vomitus may occasionally contain a few drops of fresh blood or some "coffee ground" material. In one case observed by the author a tarry stool was passed, probably owing to ulceration of the mucosa.

Other significant information in the history is loss of weight or failure to gain weight. Firmly compacted stools may be noted. In a few cases diarrhea of "starvation type" occurs.

On physical examination the patient usually is observed to be hungry looking, showing signs of dehydration and malnutrition in degrees of severity depending on the duration of the disease. He frequently has a rash around the mouth caused by the irritation of gastric juice. Gastric peristaltic waves may be seen going from left to right across the upper abdomen.

The most important physical finding is the tumor. Palpating it establishes the diagnosis quickly with-

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out need for x-ray study. In all patients observed by the author the mass was palpable. Other investigators have reported similar success.[2,7] Probably it can be done in most cases with patience, perseverance and experienced attention to details. Knowing what to expect to feel is important. Palpated through the abdominal wall, the tumor feels like an olive pit. It cannot be felt unless the infant is completely relaxed, a condition that can be induced by holding a sugar dipped nipple in his mouth. Then, supporting the infant's head and shoulders with the left hand and raising him so that his back is sloped at a 45° angle from the crib mattress in a position that enlists gravity to help bring the tumor into palpable position, the examiner palpates from the right side with the middle three fingers of the right hand.

At first palpation is done lightly, and occasionally the tumor is felt immediately. If not, the examination is conducted more firmly, both through and under the rectus muscle until the entire area is satisfactorily examined. If a mass is felt, it should be pushed away from its location, then felt for again. Palpating it a second time assures the examiner that it is indeed present and that it can only be a pyloric tumor. The tumor is easier to feel after the patient has vomited, but since waiting for him to do so may be inconvenient, a nasogastric tube (a No. 10 or 12 catheter) may be introduced and the stomach emptied if necessary. If the tumor still cannot be palpated, a second or a third examination after lapse of a few minutes may succeed. Failure to detect the tumor by repeated palpation is indication for x-ray study.

Roentgenographic demonstration of the features that are diagnostic of pyloric stenosis is rarely easy and sometimes takes a great deal of perseverance and skill. A dilated stomach, hyperperistalsis and retention of barium are suggestive, but these phenomena may also be present in other conditions. Besides, all these conditions are observable clinically before resorting to x-ray examination. However, demonstrating the narrowed pyloric canal by the "string" sign is considered diagnostic. Sometimes, when the barium enters the duodenum, it will outline the cervix-like protrusion previously mentioned.

Other causes of vomiting in this age group can be differentiated from pyloric stenosis by the absence of a palpable tumor and lack of roentgenographic evidence of pyloric stenosis. In addition, in chalasia of the cardia the persistent cardial relaxation can be demonstrated by x-ray. Pylorospasm, usually intermittent, is relieved by sedatives and antispasmodics. In cases of congenital duodenal obstruction, symptoms are usually present from birth, the vomitus is bile-stained and the x-ray shadows are characteristic. Intracranial conditions, infections and poor feeding methods as causes of infantile vomiting are usually not difficult to identify.

PREOPERATIVE PREPARATION

Pyloromyotomy is not an emergency operation. Adequate time should be taken to prepare the infant for operation. The state of hydration should be studied and chemical contents of the blood evaluated. Some babies with pyloric stenosis retain enough food and are able to compensate sufficiently to keep from losing weight for a short time; and they are ready for operation without further preparation. On the other hand, later in the course of the disease there is metabolic decompensation, and an increasing hypochloremic alkalosis will be found to have developed, as shown by an increased carbon dioxide and decreased chloride in the blood. Replacement should be carried out, using an indwelling intravenous polyethylene catheter in a major peripheral vein.[4] In most instances the amount required for a 24-hour period is 27 to 40 cc. per kg. of body weight (60 to 90 cc. of fluid per pound). Of this total, one-third should be normal saline solution. The problem will be accentuated by catabolic losses of potassium in addition to whatever amount of the ion is lost in the gastric juice. For this reason it may be necessary to add potassium chloride to the solution in the amount of 1 to 2 mEq. per 100 cc. of intravenous fluids when a reasonable urinary output has been noted. It is frequently desirable to add calcium gluconate in amount of 0.5 to 0.75 per kg. of body weight (1 to 2 cc. per pound) to prevent or to treat tetany, because alkalosis causes a decrease of ionized calcium in the blood. If the protein content of the plasma is low, serum is added in amount of 2 to 5 cc. per kg. of body weight (5 to 10 cc. per pound); and if the hemoglobin is low, whole blood is added in the same proportions as serum. The preoperative program should be interrupted and operation performed whenever the infant is in satisfactory condition. It should be altered or prolonged if a satisfactory response is not being obtained. No calculations will take the place of careful clinical observation while replacement is taking place.

OPERATION

For anesthesia the author prefers open-drop ether with plenty of oxygen run in under the mask. Atropine is the only drug used for premedication. Just before the induction of anesthesia, the stomach is emptied carefully with as large a nasogastric tube as can be easily introduced. The tube is left in place during the operation and until the infant is

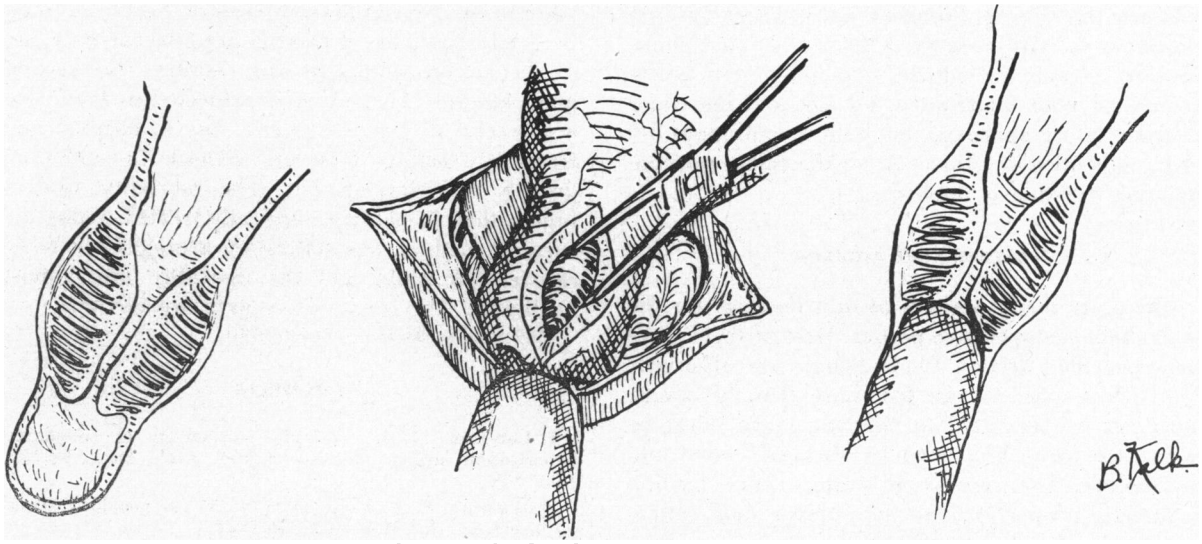

Figure 1.—*Left:* Cross-section of hypertrophied pylorus with cervix-like protrusion of circular muscle. *Center:* Position of finger reducing protrusion while performing pyloromyotomy. Note bulging mucosa. *Right:* Cross-section to diagram the effective reduction, helping to avoid perforation at the dangerous angle.

fully awake from the anesthesia. This will prevent vomiting and aspiration during and after the operation. With the stomach collapsed, delivery of the pylorus into the incision is made easier.

There are two incisions to choose between. If the tumor is palpated in a lateral position, the right upper quadrant gridiron incision serves best. The skin is incised 2 or 3 cm. below the right costal margin and parallel to it. The external oblique and the internal oblique are each spread in the direction of their fibers and retracted. The transversus and peritoneum are opened as one layer and the liver lies immediately beneath. If the "tumor" is in a more medial position, the right rectus muscle splitting incision is used. The skin is incised vertically 2 cm. to the right of the midline, starting just below the costal margin. The anterior fascia is also incised vertically and the rectus muscle spread in the direction of its fibers and retracted. The transversus muscle is usually quite well developed in this region. It too is spread in the direction of its fibers before the peritoneum is incised transversely. With this incision also the liver lies directly beneath; and it is pushed upward with the surgeon's left index finger, while the inferior edge of the wound is retracted by the assistant. A Babcock clamp is passed into the peritoneal cavity by using the back of the left index finger as a guide. It is quite easy to grasp the antrum of the stomach with the clamp and deliver it into the wound. The stomach is held with a moist sponge and so maneuvered as to deliver the pyloric "tumor." Judicious choice of incision permits carrying out this procedure with a minimum of tension on the duodenum.

The tip of the left index finger is then placed against the duodenal end of the pyloric tumor. This, in effect, pushes the protruding muscularis out of the duodenum and helps the surgeon to avoid the dangerous angle (see Figure 1). The tumor is rotated so that the more avascular anterior-superior portion presents, and the antrum is given to the assistant to hold.

An incision is made through the serosa from the pyloroduodenal junction proximally onto the antrum. It is extended somewhat deeper into the muscularis in the center or thicker portion. The muscularis fibers are then spread with a curved hemostat until the mucosa bulges up freely throughout. The most difficult and dangerous area is at the duodenal junction. Here the entire continuity of the ring must be broken without entering the lumen of the duodenum. If such an opening is inadvertently made, it must be repaired and gastric suction maintained for 24 hours after operation. Bubbles of air or bile-stained material may indicate such an opening. If there is any question, the stomach may be inflated with air through the nasogastric tube. The mucosal tear should be sutured with 5-0 chromic or arterial silk and a portion of omentum tacked over the area.

Small bleeding points in the tumor edge are ignored. The ooze is usually due to passive congestion caused by traction of the stomach into the wound. If the pyloric vein itself is divided, it may be sutured a few millimeters away from the edge with 5-0 silk.

When the pylorus is returned to the peritoneal cavity the liver comes down to its position under the incision and offers good protection against

evisceration. The peritoneum and transversus are closed as a single layer with a 3-0 continuous chromic atraumatic suture. The remaining layers are closed with interrupted 4-0 silk and the edges of the skin are approximated with a continuous 5-0 plain subcuticular stitch. A small, light, nonconstricting dressing is applied.

POSTOPERATIVE PROGRAM

There are many regimes of oral feedings for the immediate postoperative period. Most of them serve well, provided certain fundamentals are observed: The infant must not be fed more than he wants, must not be permitted to take too much, must be picked up to be fed, must be "burped" often and thoroughly. The success of postoperative feeding is directly proportional to the nursing staff's attention to the details of care.

Oral feeding may begin when the infant is fully recovered from the anesthesia, usually by four to six hours. He is offered 15 cc. of 5 per cent glucose in water every two hours until three feedings are retained. Then he is offered up to 30 cc. of a 1:5 mixture of evaporated milk and water every two hours until he has retained three feedings. The formula is then gradually increased in amount and concentration so that in 48 to 72 hours he is on a regular feeding regime and is sent home. The avererage time in hospital after the operation is three and a half days. In the first 24 hours, supplementing the diet with parenteral fluids may be desirable, particularly for infants who were in poor condition before operation, or who vomit for a day or two after operation.

The nonsurgical method of management using diet and antispasmodics has been reviewed by Rinvik[10] in the Scandinavian literature. So far as could be determined, the method is not advocated in any of the major centers of this country—for several good reasons. The infant is subjected to from several weeks' to several months' stay in hospital and frequently suffers a severe setback in nutrition, growth and development. The mortality rate is higher than in patients surgically treated. Using the surgical method, hospitalization averages five days, recovery is rapid and the mortality rate almost negligible. The economic aspects are obvious.

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